are due to alimentary toxaemia, but the poison has never been found, the symptoms have never been produced experimentally, few observers have up to the present even given an opinion on the matter, and in their extreme degree these symptoms are in my experience decidedly rare, yet we find it definitely stated, time after time, that these symptoms are due to alimentary toxaemia, but I submit that the evidence does not amount to proof, although it renders the suggestion probable. If we are not careful we shall, before we know where we are, be invoking the aid of alimentary toxaemia in the same reprehensibly loose way as gout, uric acid diathesis, and congestion of the liver have been called in to explain various vague symptoms which we do not understand.

So many conditions which cannot be called symptoms have been ascribed to intestinal stasis and consequent alimentary toxaemia, that they cannot all be examined now, but every one of them needs more careful scrutiny than it has received. Cystic disease of the breast, cancer of many parts, and duodenal ulcer, for example, have been set down to intestinal stasis, but it has not been shown that, among a large number of sufferers from any one of these, intestinal stasis is more common than among those in whom these conditions are not present. Then, intestinal stasis is commoner in women, duodenal ulcer in men, yet the first is said to cause the second. This casts so much doubt on the correctness of this suggested etiology that if there are special circumstances which can dispel the doubt they ought to be so convincingly stated that their adequacy is accepted by all. Nor have any of these conditions been induced in animals by artificially induced intestinal stasis. Then, again, we read that the "so-called Bright's disease is merely a product of intestinal stasis." There are many varieties of Bright's disease—which is meant? That being settled, let it be shown that a considerable proportion of those suffering from that variety of Bright's disease have alimentary toxaemia. This has not yet been done. Again, I see it suggested that curing alimentary toxaemia cured a case of exophthalmic goître; no such conclusion can be drawn from a single case, for exophthalmic goître is a disease which tends to get well, especially if the patient rests, and this patient rested in bed during her operation for intestinal stasis. A woman suffering from exophthalmic goître was admitted under me with typhoid fever; when she was discharged her exophthalmic goître was cured. This does not show any relationship between typhoid fever and exophthalmic goître, which was probably cured by the rest in bed.

If it is allowed that ill health is due to alimentary toxaemia caused by bacterial changes, the best treatment would be to stop the ingestion of the offending bacteria, but as we do not know what they are we cannot do this; or to give intestinal antiseptics, which experience has shown to be of limited use, although I think their uselessness has been exaggerated, for occasionally cyllin seems to do good; or to alter the medium in which the bacteria grow, which is successful when we cure children by diminishing their carbohydrates; or to give other micro-organisms which will render difficult the growth of the offending micro-organism, this is the principle of the sour milk cure, from which much was hoped but which has been disappointing; or if there is delay in emptying the intestine, to overcome this.

The last is the commonest mode of treatment, and thousands of people keep themselves in good health by natural aperient waters or some other simple aperient—either food or drug. Abdominal exercises and massage will, if properly carried out, cure many a case of constipation which has resisted all other methods, and the same may be said of a visit to a hydropathic institution or a spa which lays itself out for the treatment of this disorder. Lately some surgeons have been trying to limit the formation of poisons in the intestines by preventing delay by performing an ileo-sigmoidostomy either with or without excision of the colon. When these cases are reported we are always assured that all medical means had been adopted without benefit, but we are never told what the medical means were. It would add greatly to our knowledge if our surgical friends would give precise details of the medical treatment, the patient's statements being corroborated by the various doctors who treated her. A suspicion comes into one's mind sometimes that perhaps some surgeons do not know all the means the physician has at his command for the treatment of delayed action of the bowels.

Some have opened the appendix and washed the bowel through from the appendix to the anus; if the stasis is in the small intestine this operation will not be of much use, but the success which may follow it when performed for colitis is so brilliant that when the stasis is in the large bowel much benefit might be expected. One thing is certain—namely, that if surgical interference is ever necessary the patient for whom it is necessary is a reproach to her doctors if she has consulted any in the early periods of her illness, for the condition never ought to be allowed to advance to the stage which requires surgery, and, although no other operations may be feasible, yet short-circuiting and opening the appendix are only makeshifts, for neither removes the cause of the intestinal stasis.

It is too early to speak definitely as to the results of these short-circuiting operations. Occasionally the operation itself is fatal. I, like some others, have seen some women who are not in the least better for having had it done; on the other hand, many successes have been reported, but sometimes more than one operation has been required, and sometimes aperients are still necessary after operation. It would be a good thing if some of these cases were more fully reported; photographs before and a year after the operation would be useful, and the patients who had been operated on, whether successfully or not, might be shown before this society; and it is desirable to have the after-history of every case that has ever undergone operation, for it may be that any improvement that ensues is not permanent; this is especially important, as many of the subjects of operation are neurotic women, and we all know that the immediate effects of operation on them may be extraordinary. Further, it is unfortunate that the most frequent symptom—namely, pain—is subjective.

In this brief survey I have tried to indicate the direction which future work should take. I hope I have not been too critical; if any think so, I would beg them to remember that friendly criticism can do no harm. Our interest in alimentary toxaemia has been awakened by the fascinating suggestions of Metchnikoff and by the bold extension of them by Mr. Arbuthnot Lane, but it is an approved principle of science that the results of one worker cannot be accepted until confirmed by others. We ought, therefore, to withhold our verdict on many points until we see whether confirmation is forthcoming. In the year 600 A.D. Isidorus Hispalensis, Bishop of Seville, wrote: "For a medical man should know the *ars rhetorica* that he may be able to support with sound arguments the matters which he deals with; and also the *ars dialectica*, so that by the exercise of reason he may investigate the causes of sickness for the purposes of cure." That is true now as then.

THE BACTERIOLOGY OF THE ALIMENTARY CANAL.

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THE interests of this discussion will, I think, best be served if I devote my allotted time first to a general review of the bacterial flora of the alimentary canal and then to a consideration of the part played by bacteria in the production of alimentary toxaemia. I must leave to others the more contentious details of this large subject.

THE CONDITIONS FOR BACTERIAL GROWTH OFFERED BY THE ALIMENTARY CANAL.

These conditions are for the most part ideal. Bacteria find there an equable temperature, and the moisture they require, while the supply of foodstuffs is ample and varied. Free oxygen is largely absent in the intestine, except perhaps in certain diarrhoeal conditions; we therefore find that the habitual tenants of the gut are facultative anaërobes, and that even strict anaërobes can grow there freely. It is well known that anaërobic conditions favour profound chemical cleavages in the medium in which bacteria are growing.

While the reaction of the greater part of the alimentary canal is alkaline, and thus favourable for bacterial growth, the local acidity presented by the gastric contents is not merely unfavourable but actually destructive to the more sensitive bacteria ingested. The pneumococcus, common in the mouth, does not seem as a rule to reach the intestine, and it is believed that normal gastric acidity is a safeguard against cholera.

There is a lack of repose in the contents of the alimentary canal; its tenants are liable to daily expulsion. Too much stress must not be laid on this, for a bacterium can multiply a thousand-millionfold during the average time occupied by its transit from mouth to anus, and vast numbers must remain in the various recesses of the canal or adherent to its mucosa. Nevertheless, stasis is a fertile adjuvant to bacterial multiplication, and hence to alimentary toxaemia, whether in a dilated stomach or under the commoner form of constipation.

The Biological Characters of the Normal Flora of the Canal.

If we knew as much about the protozoan inhabitants of the alimentary canal as we do about its bacteria, we should probably find much the same laws governing both, but we know little on this subject, and I must pass the fauna by.

Of the flora of the canal we can safely affirm that it is *sui generis*. Certain groups of bacteria have specifically adapted themselves to life under the conditions just outlined, and have practically abandoned other modes of existence. Such are the bacilli of the colon group, the majority of the streptococci, and certain anaërobes. So characteristic are these of the alimentary canal of man and animals that by the universal consent of bacteriologists they are taken as indices of the faecal pollution of water, milk, earth, and other substances. They constitute the enormous majority of the normal tenants of the canal. Their biological properties have been moulded by their environment; they grow best at body temperature, they are potential anaërobes, and they show a high capacity for splitting up various foodstuffs, primarily for their own use, but incidentally to the occasional detriment of their host. Moreover, they can withstand considerable periods of desiccation, for, owing to their wholesale expulsion with the faeces, their chance of reaching a new host is thus increased.

The Numbers of Bacteria in the Alimentary Canal and their Variation in Character at Different Levels.

Our knowledge on this point is limited to the bacterial species which can grow on our ordinary culture media. It is likely that agar cultures give us an imperfect idea of the total flora, but we can control our observations by stained films, and in the case of the faeces it would appear that agar cultures, aërobic and anaërobic, yield a moderately true picture of the real state of affairs. In the crypts of the fauces and in dental cavities, on the other hand, there would seem to be many organisms which we cannot cultivate. In the following remarks I can only pretend to deal with the organisms which can readily be cultivated.

In the secretions of the healthy *buccal cavity* bacteria are present, according to Gordon, to the number of 10 to 100 millions per cubic centimetre. The most striking feature of this buccal flora is the overwhelming preponderance of streptococci, which form at least nine-tenths of the total.

In the *stomach* and *duodenum*, on the contrary, bacteria are extremely few. For in health, when the stomach empties itself properly after each meal, it is well scoured, and a large proportion of the bacteria swallowed perish during digestion.

In the *small intestine* bacterial multiplication recommences, but so long as the contents are fluid and pass rapidly along the gut, the numbers are not very high, though they increase in passing downwards. The influence of the bile, which is supposed to check intestinal putrefaction, is of a selective character, for certain of the most typical intestinal bacteria have so acclimatized themselves to it that their powers of growth are in no way impaired. Bacteriologists commonly add bile or sodium taurocholate to their culture media when studying intestinal bacteria, in order to take advantage of this selective action. We have thus learned that bacilli of the colon group can all grow well in presence of bile, and that certain of the hardier streptococci can do the same. On the contrary, the more delicate streptococci and the pneumococcus will not grow in its presence, while such organisms as *B. proteus* are much restricted in growth.

In the *caecum and colon* the conditions for bacterial multiplication become more favourable than anywhere in the alimentary tract. The intestinal contents are here mechanically delayed in order that the last remnants of nutriment may be absorbed, and the numbers of bacteria found in the faeces surpass even those of the buccal cavity. According to Houston the number per gram of normal faeces ranges between 100 and 1,000 million. Supremacy in numbers is divided between the streptococci and members of the *B. coli* group, sometimes the one and sometimes the other predominating. I have usually found *B. coli* the more numerous. In the colon, too, anaërobic bacilli begin, in lesser degree, to assume prominence, while *B. proteus* and sometimes *B. pyocyaneus* are not uncommon.

The Named Bacterial Species of the Flora of the Alimentary Canal.

This subject is one of much difficulty. At any time one is beset with doubts as to the criteria to be employed in deciding specific differences amongst bacteria; it is possible that they scarcely exist in the sense to which we are accustomed amongst higher plants and animals. And nowhere is the difficulty so great as amongst the coliform bacilli and the streptococci, which present infinite gradations in their biological characters. I cannot attempt to deal with this subject here, but must content myself with a few general statements.

The members of the *B. coli* group are roughly classified according to their fermentative reactions with certain sugars and alcohols, and in a more refined manner by their agglutination reactions. The commonest variety is that now recognized as the classical *B. coli communis*, which ferments lactose and dulcite but not saccharose. Hardly less common is its saccharose fermenting variety, whilst the various types known as *B. lactis aerogenes*, *B. acidi lactici*, etc., are frequently met with. Less common are *B. faecalis alkaligenes* and members of the Gaertner group.

As regards the *streptococci*, terminology is even more difficult. The attempts to assign provisional names to the more frequently occurring types, by Horder and myself amongst others, have met with a good deal of adverse criticism, because the sugar reactions, introduced by Gordon, upon which we chiefly relied, have been considered by others to be too variable for the purpose. This is not the place to discuss this very controversial subject, but it will be admitted that the common streptococci of the mouth and intestine are of a relatively harmless nature as compared with *Streptococcus pyogenes*. Personally I believe that a legitimate distinction may be drawn between the streptococcus most abundant in the mouth and upper air passages and a form which is common in the faeces, though it must be admitted that intermediate forms occur, so that one is probably not entitled to regard them as species. These are the types which Horder and I described as *S. salivarius* and *S. faecalis*. Even if all sugar reactions were discarded, it would still, I think, be possible to distinguish the much greater hardiness, vigour of growth, and metabolic activity of the faecal form from the feebleness of the salivary one. I have never been able to find *S. faecalis* in the buccal cavity, though the salivary type is common in the faeces.

Our knowledge of the *anaërobic bacilli* of the alimentary canal is very imperfect. The most abundant appears to be the organism which has been variously termed *B. aerogenes capsulatus*, *B. welchii*, and *B. enteritidis sporogenes*, but, with this, the ordinary putrefying anaërobe, which Klein terms *B. cadaveris sporogenes*, but which has also been described under other names, appears frequently present. In the colon, at all events, free oxygen is so far absent that these organisms, and probably other less well-known anaërobes, can flourish, and it is clear that their activity in breaking down proteins is of a very high order.

While members of the three foregoing groups of bacteria constitute the great mass of the intestinal flora, every plate culture shows that other organisms are also present in small numbers—staphylococci and sarcinae, *B. proteus*, in more than one form, and *B. pyocyaneus*. The last named organism is one with which I have repeatedly met in the intestine, and it is of interest as one which forms soluble metabolic products. As regards *B. proteus*, it would appear that its powers of decomposing protein have been a good deal exaggerated.

THE EFFECTS UPON THE HEALTHY BODY OF THE NORMAL ALIMENTARY FLORA.

It is sometimes supposed that the intestinal flora performs certain "functions" in the economy of its host. Attempts have been made by more than one observer to rear mammals or birds under germ-free conditions from birth. In many cases the creatures thus treated have not survived for many weeks, but the experiments teach little, for it is at least as likely that premature decease was due to the wholly unnatural conditions under which it was needful to keep the animals as to the absence of bacteria from the alimentary canal. I do not know of any good evidence that it is of benefit to have our intestines swarming with bacteria, many of the products of which seem harmful to us. Bacteria are not actuated by any sentiments of altruism. They take advantage of the favourable conditions offered by the alimentary canal purely for their own good; and if they do us little harm, it is because the whole race of higher animals has been evolved under these necessary conditions, perpetually present from the beginning, so that there has been ample time for the evolution of such protective mechanisms as are needful to neutralize the habitual effects of bacterial commensalism. I refer not merely to defence against bacterial invasion, but to a neutralization of their toxic products. It is conjectured that such injurious substances, absorbed from the alimentary canal, as may escape the alchemy of the liver are neutralized by the secretions of certain of the ductless glands—for example, the thyroid. There can, however, be no doubt that the intestinal flora is not without influence upon the body, even in health, for the more actively metabolic bacteria carry out protein splitting to a point beyond that which can be exercised by the ordinary digestive ferments, and of the soluble products some are known to be toxic.

THE INFLUENCE OF PATHOLOGICAL CONDITIONS UPON THE FLORA OF THE ALIMENTARY CANAL.

Owing to the extreme rapidity with which bacteria can multiply, rapid changes can readily occur in the flora of any part of the alimentary tract, as may be seen in infective conditions. Further, any part of the tract in which retention of the contents is brought about is apt to show an abnormal bacterial flora.

In the mouth and naso-pharynx infective changes are extremely common, and an invading microbe, such as the pneumococcus, *Micrococcus catarrhalis*, or *Streptococcus pyogenes*, may speedily predominate in the local flora. In chronic conditions of oral sepsis, such as dental caries or pyorrhoea alveolaris, there may be an excessive abundance of streptococci of lesser virulence.

Gastric dilatation and incompetence afford an instance of a radical change in the normal flora. The natural acidity of the gastric contents is disturbed, and in the absence of proper emptying and scouring of the viscus, yeasts, sarcinae, and other unnatural tenants come to multiply in the stagnant contents, and may form a conspicuous microscopic feature in the vomit.

Nor, in considering the bacteriology of the alimentary canal, must we forget the gall bladder, which is not rarely the seat of chronic bacterial infection. Usually such infection is by some normal inhabitant of the canal, such as *B. coli*, whereby no great change is brought about. But the infecting agent may be a foreign organism, such as the typhoid bacillus, and when this condition is persistent, as in a typhoid carrier, a more or less permanent addition is made to the flora of the intestine.

The bacterial contents of the small intestine may be profoundly modified in disease. In the rapid peristalsis of acute diarrhoeal conditions the normal inhabitants of the gut are quickly swept away and a new flora may be substituted. Thus, in Asiatic cholera, the contents of the ileum may yield almost a pure culture of the cholera vibrio.

In the colon diarrhoeal conditions may produce a similar effect, so that the liquid dejections may show a predominance of whatever microbe is at the root of the trouble, be it cholera, dysentery, or Gaertner poisoning. But from the point of view of intestinal toxaemia, constipation plays a much more important part than diarrhoea, and its effects must form an essential element in this discussion. Retention of the intestinal contents affords a fruitful field for the multiplication of the normal bacterial flora, and for the full exercise of their fermentative activities, and at the same time it gives opportunity for the absorption of whatever toxic products may be formed.

THE PART PLAYED BY BACTERIA IN THE CAUSATION OF ALIMENTARY TOXAEMIA.

Having thus shortly considered the nature and properties of the bacteria of the alimentary canal, I must address myself to the second and more difficult part of my task, that of attempting to assess the share which is borne by bacteria in the causation of poisoning from the intestines. If, in this attempt, I trespass in any way on the branches of the subject allotted to others, I must ask pardon.

In the first place I think it necessary, if this discussion is to be of any value, that we should endeavour to reach clear conceptions as to what we mean by "alimentary toxaemia." There are few terms more loosely employed, because we have so little accurate knowledge on the subject. The very terms "toxin" and "toxaemia," though they drop so easily from our lips and soothe our minds with the feeling that at least we have a name for these things even if we know nothing about them, are in truth but confessions of ignorance. The word "toxin" is a convenient one for chemical poisons of which we do not know the composition, but it might be better for clear thinking and the advance of knowledge if we were less ready to cloak our ignorance by the too common use of the term. Our hope of salvation rests with the chemists. The word "toxaemia" is equally vague, and in danger of loose usage, but it is a legitimate word if we employ it to mean the circulation in the blood of chemical poisons whereby they are enabled to attack the cells or tissues for which they have the requisite chemical affinity. By "alimentary toxaemia," therefore, I understand the absorption from the alimentary canal of chemical poisons, of known or unknown composition, in sufficient amount to cause clinical symptoms, the blood having served as the channel of distribution to the tissues which are poisoned.

This definition would exclude all those cases in which actual bacterial invasion of the blood occurs from the alimentary canal, though not those in which there is absorption of the toxic products of bacteria concerned in purely local infection of the canal. I would lay some stress on this because I think it is hardly realized how commonly slight invasions of the blood stream take place; evidence is gradually accumulating to show that the normal intestinal bacteria are constantly gaining access to the blood in trivial numbers, and as constantly destroyed by the various protective mechanisms which the body has been compelled to evolve in order to avert the danger. Only when these mechanisms are in default does serious trouble arise, but it is possible that some of the cases of local disease attributed to toxaemia are really of infective nature.

There are certain alimentary toxaemias which are clearly not of bacterial origin. We subsist on foreign proteins, hydrocarbons and carbohydrates, and of these there is abundant evidence that alien proteins may be toxic and sometimes highly so. The normal processes of digestion break down the foreign protein into somewhat simpler, though still complex, atom groups, which, after absorption, are carried to the liver, whose business it is to reconstitute the protein in a form adapted for human needs. One of the primary functions of the liver is to act as a shield against the toxicity of foreign proteins, and we have evidence that when, by the formation of an Eck's fistula, this function is eliminated, serious toxic results occur. It follows that grave hepatic disease may be the cause of a toxaemia, partly, at least, alimentary in origin, and instances of this, in acute yellow atrophy and some puerperal eclampsias, will at once occur to the mind.

The bacteria in the alimentary canal, in virtue of the high fermentative activity which many of them possess, carry out protein cleavage to a point beyond that which the body demands for its own nutritional needs, and certain of the products which thus arise are actually harmful to the host. But this has always been so, ever since bacteria took up their residence in the alimentary canal of animals, and it has been necessary for animals to evolve some means of chemical antagonism to neutralize this harmful effect. We are still in comparative ignorance of the precise chemical mechanism by which this permanent menace is met in health, but that it is so met is certain, for in health we remain untouched. It is conjectured that one of the functions of the thyroid gland is to deal with this matter. A number of observations by different workers have shown that the activity of this gland is excited by the presence of bacterial toxins, and it has been asserted by certain observers that thyroidectomized animals are abnormally susceptible to infective processes. It is possible that the antitoxic functions of the thyroid extend to chemical poisons other than bacterial toxins. Be this as it may, it is clear that the body in some way compensates the ordinary activities of bacteria in the intestinal tract, so that no harm results. Only when such bacterial decompositions are excessive, and when faecal retention affords unusual opportunities for the absorption of the products, are symptoms manifested.

Thanks to the labours of chemists, our knowledge of the protein decomposition products is fairly extensive, though our knowledge as to their toxicity is incomplete. Most of them appear to be of feeble toxic powers, but it must not too hastily be assumed from animal experiment that such substances as, for example, indol are so harmless as they would appear. I am very far from agreeing with the recent hypothesis suggested by Metchnikoff as to the rôle of indol and allied substances in the causation of arterial degeneration, for the evidence put forward seems to be wholly insufficient. But we can to some extent measure the absorption of indol by the excretion of indican in the urine and correlate its excess with clinical phenomena. The excessive indicanuria in conditions of mental depression and melancholia, for a knowledge of which I have to thank Mr. Mackenzie Wallis, may be of great pathological significance, and may outweigh any negative evidence derived from animal experiment.

There is also a possibility, for the suggestion of which I again owe my thanks to Mr. Wallis, that excessive bacterial activity in the intestine may have its negative as well as its positive influence. Thus, still to take indol as an instance, it is known that this substance is derived from the tryptophane element in proteins. If tryptophane is an important element in tissue nutrition, it may be that excessive splitting up of this substance in the intestine by bacteria may lead to tissue starvation in an important detail. An element in a supposed alimentary toxaemia may possibly be due not so much to the positively toxic action of the derivative as to the absence of the needful substance from which it has been derived. I must leave such considerations to those who follow in this discussion.

Bacteria, however, may do more than provoke merely fermentative changes in the medium in which they are growing. They produce, in virtue of their own intrinsic metabolism, substances of unknown chemical composition which are deleterious to their hosts, and which we must be content to term "toxins" in th[illegible]f more exact knowledge. It is established t[illegible]a very few—bacteria produce soluble toxins which pass out into the surrounding medium without any disintegration of the bacterial body. These are the pre-eminently "toxic" bacteria, of which the diphtheria and tetanus bacilli are the commonest examples, and they lie outside our present subject. Indeed, almost the only essentially toxic bacterium which could be dragged into the discussion is van Ermengem's *B. botulinus*, present in certain ham poisonings, though *B. pyocyaneus* might perhaps also be included. But the great majority of pathogenic bacteria, and practically all those which are normally saprophytic in the alimentary canal, form no soluble toxins; so far as they possess toxic properties, these reside in the bacterial protoplasm itself and are liberated only on its dissolution—so-called "endotoxins."

Bacteria are, of course, constantly undergoing dissolution in the alimentary canal, and one cannot dispute the possibility of harmful effects from such endotoxins on absorption. But we are in almost complete ignorance of the extent to which alimentary toxaemia rests on such a basis. So far as concerns the normal intestinal flora, I should be disposed to believe that poisoning by bacterial endotoxins plays a comparatively negligible part in the process. As soon as evidence is brought forward showing this to be the case I shall be ready to accept it, but so far there is none, and it is probable that the endotoxins of most of the normal inhabitants of the alimentary canal are of very feeble virulence, though those of the colon bacillus may perhaps prove of some importance. This does not, of course, apply to cases of infective enteritis, in which the absorption of bacterial toxins plays a large part in the production of the constitutional symptoms.

To sum up what I have said as to the part played by bacteria in alimentary toxaemia, it seems to me reasonable to lay down the following propositions:

1. In an important group of cases the poisoning is by foreign proteins, as such, the defect lying in the liver, which ought to shield the body from their effects. With this bacteria have, as a rule, nothing to do.
2. The main effect of bacterial activity in the production of alimentary toxaemia lies in their ability to carry out protein cleavages beyond the capacity of the ordinary digestive ferments, with the formation of products which, when in excess, the body is unable to neutralize.
3. There is at present little evidence that true toxins, derived from the ordinary flora of the gut, soluble or intracellular, play much part in alimentary toxaemia.

ALIMENTARY TOXAEMIA: ITS SYMPTOMS AND TREATMENT.

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THE doctrine of intestinal autointoxication was originated by Bouchard, and has formed the foundation for far-reaching speculations. There is little evidence that the normal products of intestinal fermentation and putrefaction are capable of producing disease, and if acetic and butyric acids, indol, skatol, and the like are harmful their effects must not be exaggerated. Sulphuretted hydrogen is only present normally in traces, and much larger quantities may be injected into the rectum without hurt, although admittedly this gas is more harmful when formed in the small intestine or the stomach. Ammonia is converted into harmless urea, while volatile fatty acids and acetic and butyric acids only produce local stimulation, even when present in quantity. Acetone, diacetic acid, and beta-oxybutyric acid are present in the urine, together with various symptoms of disturbed digestion, but this does not prove that the symptoms are due to them. The first two are found in the urine of starving animals with no digestive disturbances, and merely indicate the breaking down of tissue protein. Ptomaines may be present in excess in intestinal contents without any toxaemic results, as, for example, in cystinuria. On the other hand, there is a widespread belief, not without evidence in its favour, that many diseases depend upon the absorption of poisons from the intestine, either introduced with food or formed from it by the fermentative or putrefactive processes in the alimentary tract.

FOOD POISONS.

In former days food poisoning referred to the accidents that result from the ingestion of substances rendered harmful by the poisons contained in them, but modern research has added to these the effects of microbes and the toxins due to their activity. These last are exclusively produced from animal food; the danger of vegetable food depends solely upon its natural poisons or its accidental contamination by microbes in the water with which it has been washed or in the dust lying on its surface.